sinking to the bottom of the jar, but the second change of the solution has remained clear while the liver remains dark green in colour.

A considerable number of old specimens previously mounted in the Kaiserling sodium acetate glycerol preservative solution, which had undergone repeated discoloration after the fluid had been changed, have now been remounted in liquid paraffin. In the great majority there has been no further loss of pigment, but in one specimen of spinal column the blood pigment has continued to leak into the liquid paraffin. In a few a small amount of pigment has formed a thin sediment at the bottom of the jar.

It seems that the use of liquid paraffin as a final preservative solution in which pathological material is mounted will obviate much of the discoloration of the mounting fluid that so detracts from the appearance of pathological specimens in medical museums. Not only will the specimen look more lifelike, but there will also be a considerable reduction of expense when less satisfactory Kaiserling preservative fluid, which has to be changed repeatedly, is replaced by the essentially inert, non-reactive liquid paraffin. It should be noted that 25 litres of liquid paraffin (British Drug Houses) cost £23.93 as opposed to £35.67 for a similar amount of glycerol (Fisons).

This method has been tried on fresh specimens for only one year, which is admittedly too short a time for final evaluation; many another promising technique has been proved wanting by the passage of time. However, we first tried liquid paraffin eight years ago on an enormous old spleen with chronic myeloid leukaemia, and the organ is still in a state of good preservation.

Reference

Letters to the Editor

A comparison of viral transport media

Following the report of Chaniot et al. (1974) that storage survival of some respiratory viruses was increased when HEPES rather than bicarbonate buffer was used in media, we report an initial comparison of the survival of herpes simplex virus (HSV) at room temperature in viral transport media (VTM) comprising Hanks' balanced salt solution (HBSS) and 0-2% bovine albumin, buffered (pH 7.2) with HEPES and bicarbonate respectively. When bottles of each were inoculated with a field strain of HSV to a concentration of 63 TCD<sub>50</sub>/ml, viable virus was detected for 29 days in the former and for 21 days in the latter. Increasing the bovine albumin concentration to 1% in the bicarbonate VTM resulted in virus detection for 17 days, and when 0-425% lactalbumin hydrolysate was also included, for 15 days. Comparisons of the survival of respiratory syncytial virus (320 TCD<sub>50</sub>/ml) in HEPES VTM and one with HBSS, 1% bovine albumin and 0-425% lactalbumin hydrolysate showed survival of viable virus for at least 13 days in the former and for only three days in the latter.

These initial findings suggest that (a) the use of HEPES buffer results in longer virus viability, and (b) neither increasing the bovine albumin content nor adding lactalbumin hydrolysate significantly prolongs survival of labile viruses in bicarbonate buffered VTM. The increased cost of using HEPES instead of bicarbonate buffer is 33p/litre.

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Reference

Diagnosing thalassaemia trait from platelet count and England's discriminant function

We are very interested in Hegde's recent paper (Hegde et al., 1977) reporting that a thalassaemia trait may be shown using England's discriminant function (DF): DF = MCV - (Hb x 5) - RBC - 34 (1), calculated from indices obtained on a Coulter Model S, if the levels of Hb A<sub>2</sub> and Hb F are within normal limits.

It appears, however, that this function is unable to distinguish between all cases of thalassaemia. England (England et al., 1973; England and Fraser, 1973) found a negative value of the DF to be significant for thalassaemia trait and a positive one for iron deficiency; in Hegde's cases, the DF is positive in 7 of 13 confirmed thalassaemia traits, in 19 of 57 suspected thalassaemia traits, and in 3 of 269 beta thalassaemia traits, that is, in about 30% of the heterozygous thalassaemias.

We reported (Seigneurin et al., 1977) that the thrombocyte blood count used
with the DF is able to separate thalassaemia trait from iron deficiency; 50 patients have an MCV of less than 75 fl; a high platelet count (more than $400 \times 10^9/\text{l}$) is found only in the cases with iron deficiency; when the platelet count is normal or low, the discriminant function differentiates the iron deficiency (positive DF) from the thalassaemia trait (negative DF). So we were able to identify 26 of 28 cases with iron deficiency and 17 of 19 cases with $\beta$ thalassaemia trait.

In conclusion, the platelet count associated with the discriminant function may serve as a useful screening tool to detect the majority of patients heterozygous for thalassaemia or deficient in iron.

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A case of small-cell Sézary syndrome with null-cell features (Goldstone et al., *Journal of Clinical Pathology*, 1976, 29, 848)

This case was originally investigated and treated at the Royal West Sussex Hospital, Chichester. He presented in December 1974 with a history of a rash on the thighs. A skin biopsy was reported as the plaque stage of mycosis fungoides (Dr Knowles). He symptomatically improved on therapy with Dimotane and Dermonate (1 in 3) but soon relapsed and was admitted to hospital in February 1975. At this time the rash (which was itchy) was widespread over the trunk (back and front) and legs. A second biopsy was again reported as typical of mycosis fungoides. There was no splenomegaly nor lymphadenopathy. Haematological investigation showed the white count to be raised at $29 \times 10^9/\text{l}$ with $70\%$ small, mature-looking lymphocytes. Haemoglobin level and platelet counts were normal. Bone marrow aspiration did not show any lymphocytic marrow infiltration.

Histological examination of a normal sized lymph node, removed from the groin, showed the presence of a well-differentiated, diffuse, lymphocytic lymphoma.

The surface characteristics of the peripheral blood lymphocytes were studied in March 1975; $70\%$ were null cells, $20\%$ T cells, and $10\%$ B cells. Transmission electron microscopy showed the cells to be Sézary cells. Over the next three months the skin condition remained much the same but the WBC count rose progressively until, by the end of May, it had reached $58 \times 10^9/\text{l}$ with mature lymphocytes predominating. On 29 May 1975 treatment was started with chlorambucil, 6 mg daily. The total WBC dropped steadily over the next eight weeks and, towards the end of this time, the skin condition suddenly improved. Just before this occurred arrangements had been made for the patient to be transferred to Cambridge for consideration of whole body irradiation.

Although this additional information does not affect the conclusions of Goldstone et al., it does illustrate the diagnostic problems associated with this bizarre disorder. We feel your readers will find it interesting.

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Occurrence of e antigen in acute hepatitis B

The recent paper by Gibson and Ruparelia (*.Journal of Clinical Pathology*, 1977, 30, 925-927) suggests conclusions which are quite unjustified by its evidence. Of 44 patients with hepatitis B, only six were found e antigen positive, and only two became HBsAg carriers (why substitute $13\%$ and $4.6\%$ for integers in the summary?). If presence of e antigen and becoming a carrier of HBsAg were independent, the probability of their coincidence in this group would be about 0-006, so the finding reported proves nothing. In addition, the authors acknowledge that they may have failed to detect e antigen in some of their patients and did not do so in two of three who eventually formed anti-e. Their conclusions about the duration of HBs antigenaemia are also open to criticism for the same reasons.

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The author comments as follows:

In reply to Mr Davey's letter of 29 November 1977, I should like to make the following comments: the results in our paper are straightforward and although no firm conclusions are reached, those that are, are self-evident. No claim of independence is made. Of six cases where e antigen was found, none became a carrier of HBsAg, indicating that the presence of e early in the course of acute HBsAg is not necessarily of prognostic value. Unfortunately, blood samples from the early acute phase of illness are difficult to obtain, and of 90 cases of acute hepatitis studied, only 44 satisfied the necessary criteria to be included (ie, first serum sample taken within one month of jaundice onset and the final sample cleared of HBsAg or when it became evident that the carrier state had developed). Those not included were patients whose serum samples were taken very late during the HBs antigenaemia and these were all e negative. The importance of this report is that in cases where blood samples were available from within one week of onset of jaundice, e was detected in these first specimens (but not in the following specimens) in 6 of 15 cases, while in the remaining 29 cases where the first specimen was obtained following one week of the onset of jaundice, e was not detected in any of the samples. The difference between these two groups is very significant.

Berquist et al. (1976) and Frössner et al. (1977) have reported similar results and reached similar conclusions. Therefore,